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ABSTRACTS

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Hemoglobin at High Altitudes.—The position of the oxygen dissociation curve has been determined in arterial blood obtained from (*a*) 17 healthy adult men living at sea level; (*b*) 12 Indian native residents of Morococha (Peru), at an altitude of 4,540 meters (14,890 ft.). The same determination has been repeated in 12 subjects of the first group within the first two hours after arrival at the high altitude, and in 8 men of the second group also within the first two hours after arrival at sea level. The results have been compared with those obtained in previous investigations. There seems to be enough evidence to conclude that in humans, new-comers or residents, at altitudes of about 4,000 meters or higher, there is no increased affinity of the blood hemoglobin for oxygen. On the contrary, the findings indicate a slight tendency toward a lower affinity, both at arterial pH and a standard pH of 7.40, especially in the native residents. This right shift in the oxygen dissociation curve at high altitudes may be interpreted as a favorable compensatory adjustment to the low pressure environment. (Authors' summary.)—*The Affinity of Haemoglobin for Oxygen at Sea Level and at High Altitudes, H. Aste-Salazar & A. Hurtado, Am. J. Physiol., December, 1944, 142: 733.*—(*G. C. Leiner*)

Pulmonary Syphilis.—This is a detailed review on pulmonary syphilis from a pathological point of view, based on the observation of a series of lungs with acquired or congenital syphilitic lesions. The division of syphilis into three periods is compared with the known scheme of Ranke concerning tuberculosis. The division in the case of syphilis is not based on an immunological conception and the lesions in tertiary syphilis have a definite pattern, while in the tertiary tuberculous lesions an anatomic polymorphism prevails; besides these differences, the division in the case of syphilis responds to practical needs and is adequate, while Ranke's scheme, although the best attempt as yet made to obtain a global vision of tuberculosis, is still widely debated and insufficient for practical use. The microscopic examination of syphilitic specimens has two objectives: the demonstration of the treponema in the fresh material with the usual techniques and the histological study of the tissue changes. For the latter, the fixation of the lung by 10 per cent formol solution, before the opening of the chest, is advised. Postmortem X-ray examination is also a useful technique. The participation of the bronchi in form of gummous bronchitis or bronchiolitis obliterans was evident in all cases. Peribronchial lesions were also present. The possible rôle of syphilis in the genesis of bronchiectasis and also the possible relationship between congenital cystic lung disease and syphilis are pointed out. The part played by the parenchyma in pulmonary syphilis is not clear. Nodular alveolar lesions were seen only exceptionally; more often bronchopneumonic alterations were found, either of the nonspecific type, with cellular and albuminous exudate of the acute type, or specific infiltration with mononuclear cells. The alveolitis was mostly accompanied by interstitial pneumonia. The interstitial tissue is the site of the most important and extensive

alterations. Vascular lesions of all layers and types were found in the material. Among the sequelae of the syphilitic process, cavities are rare; more frequent is sclerosis of different types, from simple linear sclerosis to extensive indurative syphilis. Various changes can be found around syphilitic lesions, such as edema, congestion, interstitial hemorrhages, pigment deposits, emphysema, atelectasis, carnification. In 2 cases exudative pleurisy was found, without any character of specificity. The gross anatomic appearance of pulmonary syphilis is rarely that of a single syphilitic gumma. Diffuse or modular gummata are frequent in the pneumonic or bronchopneumonic type of lesions. When the gross appearance of the lesions is that of sclerosis, only the presence of micro-gummata can confirm the diagnosis. Sometimes suppuration or gangrena complicates the picture. There is no agreement about the mutual influence of coexistent syphilis and tuberculosis, and the limited number of observations in this review brings no conclusive evidence in either sense. Congenital pulmonary syphilis is characterized by the abundance of treponemata that are easily demonstrated. The gross anatomic appearance is that of the so-called "pneumonia alba." In the microscopic picture there are alveolar infiltration, interstitial lesions and a pseudoglandular aspect of the lung, corresponding to the early phase in the embryonal development of the lung. This thorough study proves once more the difficulty of the anatomopathological diagnosis of pulmonary syphilis and it emphasizes the diagnostic value of the demonstration of the treponema in questionable specimens.—*Las sifilis pulmonar, A. L. Matteo, Arch. tisiol. y pneumol., 1943, 2: 209.*—(*L. Molnar*)

Pulmonary Syphilis.—A syphilitic process of the lungs and airways can be easily mistaken for tuberculosis, bronchopulmonary gangrene, neoplasm, mycosis or bronchiectasis. Repeated attacks of "influenza" or pneumonia, as well as signs of diminished aeration of the lungs should always lead to a suspicion of syphilis. The most frequent location of pulmonary syphilis is in the left lower lobe. The authors present a case that had been diagnosed and treated for many months as a primary neoplasm of the lungs. When this patient was first seen he presented an almost complete aphonia and a tracheo-cutaneous fistula was found in the region of the cricoid cartilage, which was completely destroyed. The serological examinations were always negative and only after a diagnostic injection of arsphenamine did the Wassermann test turn positive. The symptoms subsided on specific antiluetic treatment.—*Sifiloma pulmonar con localizacion laringea, J. M. Remolar, V. Thompson & G. Caputo, Rev. Asoc. méd. argent., May 30, 1944, 58: 340.*—(*W. Swienty*)

Pulmonary Mycosis.—This article is intended as a review and summary of the literature regarding yeast infections of man, especially of the lungs. It is believed that, from a practical standpoint, there is sufficient similarity in the lesions produced that all yeast infections may be discussed as a single entity, any variation being chiefly one of degree of virulence. Practically no regions are exempt, though some have a higher incidence, such as coccidioidomycosis in the San Joaquin Valley. Susceptibility varies little with race, color or age, but depends mostly upon geographical location. Neither does there seem to be any occupational prevalence. In spite of the frequent number of cases of blastomycosis of the skin encountered among persons raising or handling cattle, the incidence of pulmonary mycosis is no higher than in any other occupation, though by contrast a newcomer to the San Joaquin Valley may contract a mycotic infection while the natives have little trouble. The spores of yeast are very resistant to heat, cold and drying, but grow in a slightly acid medium. Since the body fluids are slightly alkaline, it is possible that some other disease may temporarily change the pH so that organisms may begin to grow. Primary infection occurs most frequently through the respiratory tract, possibly bronchial ulceration, allowing the yeast to gain a foothold. Yeasts do not seem to produce much defense

mechanism by the body. Extension may occur from the yeast alone, in conjunction with some infection, or areas of bronchopneumonia may develop as the result of an associated disease taking advantage of the injury done by the yeast. At some stage in the spread, a large number of organisms must get into the blood or lymph stream of the more virulent infections producing new areas of infection all through the body which grow rapidly and are of high degree of toxicity, especially in coccidioidal and torula infections. Probably most yeast infections never get beyond the bronchi. Microscopically the lesion of a mycotic infection is a tubercle-like granuloma. Complicated by other infections, it will also show the microscopic cell structure of these infections. The symptoms of pulmonary mycosis vary in degree and type depending upon the virulence of the infection, extent and rapidity of extension. In mild cases the symptoms are those of any pulmonary infection, a chronic cough, often productive, being the most common symptom. In severe cases there may be fever, hemoptysis and other evidence of pulmonary disease, a foul nauseating sputum being the only sign suggestive of a fungus infection. In disseminating disease, the symptoms depend on the organ involved. Physical examination often reveals few abnormal findings, and these are present also in tuberculosis and other pulmonary diseases. In some cases the physical findings are extensive, but often they do not indicate any severe changes, and the clinician may be surprised at the extensive disease shown by the X-ray film. While the clinician is the first to recognize the pulmonary disease, the roentgenologist can often suggest its nature, and the pathologist or bacteriologist must make the final diagnosis by actual demonstration of the fungus itself. Probably the earliest roentgen findings are only increased marking of the bronchovascular tree and some irregular peribronchial infiltration which may be unilateral. No enlargement of the lymph nodes was found in any of the cases studied. Later patchy areas of density appear in the parenchyma, miliary in size or as large nodules, which may increase, coalesce and break down into abscesses. The lesions may gradually extend toward the periphery, eventually producing pleural thickening. Another type of involvement may show faint, fuzzy shadows along the bronchovascular tree giving a ground glass appearance. Advanced cases may present discrete nodules in the pulmonary parenchyma quite dense and not sharply demarcated, varying in size, isolated or conglomerate. During convalescence these lesions may melt away like snow, and extensive changes take place in a few weeks as compared to the slow change seen in tuberculosis. Some do not clear entirely but leave areas of fibrosis and chronic cavities. Tuberculosis frequently does develop in a mycotic lung, and it would be difficult to tell which disease occurred first. The tendency to attach little significance to the presence of yeast once a tubercle bacillus is demonstrated may be a definite mistake for the yeast may play an active part in the lung, and little improvement may be expected until the fungus infection is largely eradicated. X-ray differentiation from tuberculosis is not at all reliable. Other diseases from which mycotic infection must be differentiated are sarcoidosis, cancer metastases, bronchopneumonia, atypical and virus pneumonia and the nodular stage of pneumonoconiosis. The most widely used and successful treatment appears to be the use of iodides and X-ray. Increasing doses of potassium iodide by mouth and sodium iodide intravenously, even in the presence of tuberculosis, and X-ray radiation once or twice a week over the chest and skin lesions, using not over 100 r per area treated, are used. Some authors believe that the patient must be desensitized to the yeast before receiving iodides in the treatment of moniliasis and blastomycosis. Lucas vaccine 0.1 cc. twice daily increasing to 0.8 cc. or immune rabbit serum may be used. Cases are reported demonstrating some of the difficulties encountered in evaluating the importance of yeast in the sputum, where there may be a pure infection, a secondary infection which assumes a primary rôle in the extension of the original disease, the secondary disease or both.

The authors feel that yeast infections are more prevalent than ordinarily thought.—*Clinical and Radiological Studies of Pulmonary Mycosis, W. A. Johnston & J. Heydemann, Radiology, July, 1944, 43: 1.—(G. F. Mitchell)*

Fungus Diseases of Chest.—A group of proved cases of infection of the lungs due to fungus infection are presented. In approximately one-fourth of these cases, there were fungus infections elsewhere in the body. Such extrapulmonary lesions are an aid in the differential diagnosis of the pulmonary lesion. Twenty-four cases of coccidioidomycosis were diagnosed by sputum culture, positive skin tests or blood agglutination tests. The X-ray evidence of these cases varied greatly. The most frequent lesion was a solitary nodose area of infiltration that later broke down leaving a thin-walled cavity, but at times the nodose lesions were scattered throughout the lung so that they gave the appearance of metastatic carcinoma. In other cases they closely resembled tuberculous lesions involving one or both apices or midlung field. About half of the patients showed slight enlargement of the hilar lymph nodes. During the time of observation, the infiltrative lesions cleared slowly with only slight change in the size of the cavities. The histories were indefinite, but all were from southwestern United States. In most there was little clinical evidence of disease at the time of admission to the hospital. The residual cyst-like cavities reported by Winn and Johnson were demonstrated in these cases, and while no lipiodol studies were made, a number would undoubtedly also show bronchiectasis. One patient had a positive coccidioidin skin test and negative tuberculin test, and while there was no history of infection, it is assumed that the parenchymal scar was due to coccidioidomycosis. In actinomycosis there is an early extension from the lung into the pleura, and the latter involvement may obscure the underlying pulmonary lesions. Nine of the cases observed showed early or late pleural involvement. The lungs showed infiltration which could not be differentiated from tuberculosis; one or both lungs were involved and abscess formation usually was early and often obscured by the pleural lesions. Cases coming to autopsy revealed extensive pleural and pulmonary involvement usually extending through the diaphragm to the abdominal viscera. In the group of cases classified as aspergillus infections, similar radiographic finds were present, X-ray films revealing a soft, irregular, peribronchial or parenchymal infiltration and often enlargement of the hilar lymph nodes. All showed rapid clearing with iodide therapy, and one cleared without therapy on bed-rest. In such cases, if repeated sputum tests for tubercle bacilli are negative, one may be justified in suggesting iodide therapy and basing the diagnosis on the response to treatment. The author reports examining roentgenograms in which multiple irregular areas of calcification are seen in the parenchyma and hilar nodes with or without a negative tuberculin test. This type of calcification is regarded as the residual of a healed fungus infection. *Monilia albicans* is commonly harbored by man and, while it frequently is a secondary invader in pulmonary disease, it may also produce primary pulmonary lesions. As seen on X-ray films there is a diffuse bilateral and fairly symmetrical, peribronchial infiltration. One case broke down with resultant cavity formation. If treated the lesion resolves rapidly with residual fibrosis. Torulosis and blastomycosis are rare in man and usually produce parenchymal lesions similar to those seen in tuberculosis. One case observed over a period of six years and diagnosed as tuberculosis despite repeated negative sputum tests died of a miliary spread and was found at autopsy to have blastomycosis. Thus, while fungus infections of the lung are not common, they do occur, and it is important that an early diagnosis be made before extensive damage occurs as many of the lesions will respond to therapy. A wide variety of lesions as seen roentgenologically may be found, but if familiar to the radiologist much help may be given in the diagnosis. Final diagnosis rests upon isolation of the fungus as well as absence of other etiological factors. The lesions most

often resemble a tuberculous involvement. Therefore, any lesion having the appearance of tuberculosis in which no tubercle bacilli can be found should be studied carefully to rule out a fungus infection, and where no definite organism can be isolated a therapeutic test with iodides may be instituted, and the final diagnosis based upon the response. It is possible that many of us will be seeing cases of fungus infection due to coccidioides as men are returned from army camps, and it is important that the true nature of the lesion be recognized.—*Fungus Disease of the Chest, V. L. Peterson, Radiology, July, 1944, 43: 14.*—(*G. F. Mitchell*)

Coccidioidomycosis.—Fourteen patients with proved diagnosis of coccidioidomycosis and 30 patients in whom the diagnosis was suspected but not confirmed were observed. Of the 14 patients, 10 had primary benign and 4 had progressive disseminated coccidioidomycosis. The history of exposure in an area where the disease is endemic is of importance for the diagnosis. All patients had been in such areas. In 4 patients the onset was with the clinical picture of pneumonia, in 2 like pneumonia with pleural effusion, in 5 insidious with cough, weakness and slight fever, in 3 the disease was discovered by repeated routine cutaneous tests. Usual symptoms were cough, pain in the chest; however, there are no symptoms or signs pathognomonic of primary chronic coccidioidal infection. Reaction to intracutaneous injection of coccidioidin, in dilutions of 1:1000 and 1:100, probably indicates that the patient has or has had at some time in the past a coccidioidal infection. A negative coccidioidin reaction generally excludes coccidioidomycosis; but disseminated coccidioidomycosis may have a negative phase. Examination by direct smear is not reliable; culture and inoculation into a guinea pig or mouse should always be done. Serological tests are of help for establishing the diagnosis; negative serological tests speak against the diagnosis of disseminated coccidioidomycosis, but not against primary infection. Sometimes it is advisable to make cultures from material obtained by biopsy. X-ray findings may be simple hilar thickenings, pneumonic infiltrations, massive effusions; solitary, thin-walled cavities with little surrounding reaction are characteristic. Treatment is symptomatic. For the pulmonary lesions bed-rest is advised. Immobilization of the lung by a lead shot bag on the chest seemed to be of benefit. Phrenicolysis was done in 2 cases with good effect. Chemotherapy is of no value.—*Diagnosis and Treatment of Chronic Coccidioidomycosis, E. J. Denenholz & G. Cheney, Arch. Ind. Med., November, 1944, 74: 311.*—(*G. C. Leiner*)

Coccidioidomycosis.—Pulmonary lesions in 75 cases of primary coccidioidomycosis were studied. The clinical symptoms of the disease consist of dry cough, chest pain, malaise, fever, weight loss and night sweats. As a rule, recovery occurs in two to six weeks; rarely, disseminated granulomatosis develops and, then, the outcome is fatal. Recent exposure to dust containing the chlamydospores of the fungus, no history of previous exposure, a certain incubation period, positive intracutaneous coccidioidin reaction and positive complement fixation were the required diagnostic criteria in the cases studied. Twenty-four per cent of the cases exhibited increased hilar densities only, representing subacute inflammatory changes in the mucosa and submucosa of the larger bronchi and in the lymphatics. In some of these cases circumscribed lymphadenopathic shadows were seen; the latter are probably explained by more marked involvement of lymphatic structures. In 38.7 per cent of the cases fan-shaped densities were seen extending from the hilum outward, and these persisted for 15 to 90 days; pathologically, they signify involvement of the tertiary bronchi and bronchioles, together with proliferative and cellular peribronchial infiltration. Lobular and sublobular exudates, representing further extension into the pulmonary parenchyma, were found in 26.6 per cent; resolution was slow, averaging 37 days. In 3 cases (4 per cent) there was cavitation, lasting 60 days in one case, 95

days in the other, and persisting in the form of a thin walled cyst in the third. In one case intermittent ballooning of the cavity was observed. Two cases (2.7 per cent) had massive pleural effusion which resulted in considerable pleural thickening. Slight to moderate pleuritis was seen in 7 other cases. Among the above roentgenological types increase in the hilar shadow was the most constant finding. Delayed but complete resolution is considered characteristic of the disease.—*Roentgenological Types of Pulmonary Lesions in Primary Coccidioidomycosis, J. R. Colburn, Am. J. Roentgenol., January, 1944, 51: 1.*—(*P. Lowy*)

Blastomycosis of Lung.—Ayerza coined the expression, "Cardiaco Negro," for a syndrome of cardiac insufficiency and severe impairment of the pulmonary circulation which causes typical deep black cyanosis. Various etiological factors may contribute to cause this syndrome. A case with unusual etiology is presented. The patient who entered the hospital in the terminal stage of the disease presented an extensive cyanosis, severe dyspnea, edema, clubbing of fingers and an emphysematous thorax. The heart sounds were normal but for an accentuation of the second pulmonary sound. Over the base of the lungs some coarse râles were found. In the nasal septum a scar resulting from blastomycotic ulceration was found which he had had for two years and which had healed on administration of sulfathiazole. X-ray examination showed generalized pulmonary fibrosis, especially of the left lower lobe, with pleural effusion and an enlargement of the entire heart, but chiefly of the left side. The autopsy revealed a riding thrombus of the bifurcation of the pulmonary artery, sclerosis of the aorta and numerous nodes with all the morphologic characteristics of blastomycosis throughout the entire lung. Similar lesions were found in the cortex of the adrenal glands. Three principal types of blastomycosis have to be considered: (*1*) the micoderma or cryptococcus (Gilchrist and Stokes), (*2*) the coccidioides (Gilchrist and Rixford), (*3*) the paracoccidioides brasiliensis. From the original lesion in nose and septum the lungs and the suprarenals were infected either by the lymphatics or, more probably, by the bloodstream.—*Blastomicose do pulmao e das capsulas suprarrenais: Sindromo de Cardiaco Negro, A. de Almeida Prado, Rev. Asoc. méd. argent., September 30, 1944, 58: 789.*—(*W. Swienty*)

Actinomycosis.—A case of generalized actinomycosis is reported. The first manifestation was a painful swelling in the left axilla, from this 30 cc. of thick white pus was removed but no organisms were found. Ten days later the axilla was again incised, a biopsy was done, the pus was reëxamined and a diagnosis of actinomycosis was made. All modern methods of treatment were employed without appreciably affecting the progress of the disease. Four weeks after admission an enlargement resembling a left inguinal adenitis appeared and one month after admission the presence of pulmonary findings was noted; chest films at this time revealed multiple deposits distributed evenly through the parenchyma of both lungs although a chest film eleven days previously was normal. Death occurred seven weeks after admission; the final pathological picture resembled in many respects the end result in miliary tuberculosis. The finding of inflammatory tissue in the thoracic duct suggests this origin for the hematogenous spread.—*Actinomycosis: Report of a Case with Miliary Chest Lesions, A. M. Harris & J. B. Priestley, J. Lab. & Clin. Med., August, 1944, 29: 815.*—(*F. G. Petrik*)

Actinomycosis and Diabetes.—Generalized actinomycosis is an infrequent disease in Uruguay. In the past several years only 2 deaths have been reported to the register of vital statistics. The author presents one case report. The patient, a 51 year old male, came under observation because of a lung abscess which resolved completely after sulfonamide and arsenical therapy. Two years later diabetes appeared, controlled at first by diet and later with 20 to 30 units of insulin. Three months later a cold abscess of the dorsal

spine slowly developed, followed by a pulmonary lesion in the left lower lobe, a pleural effusion and chest wall abscess. Actinomyces identified as *Cohnistreptothrix Israeli* were cultured on two occasions from the pus of the chest wall abscess. A week after the first aspiration of pus the patient died. Because of the marked dyspnea and tachycardia the possibility of a mycotic cerebral lesion was considered. There was no autopsy.—*Actinomicosis generalizada de tipo septicopiohemico en enfermo diabetico, R. Rimini, Rev. de tuberc. d. Uruguay, No. 1, 1944, 12: 39.*—(*R. Kegel*)

Atypical Pneumonia.—The fairly typical clinical course and distinctive X-ray findings have given this relatively new disease a place in its own right. It is a disease of young people with a predilection for males, but any age group may be affected. The onset is with signs and symptoms of an upper respiratory infection. X-ray findings are prominent early and increase for a few days, whereas physical signs in the chest are wanting or negligible. The lower lobes are predominantly affected, and especially the paracardiac region is frequently involved. The sputum shows no predominance of any single organism. Occasionally pneumococci are found which cannot be typed in the majority of cases. The white count is slightly elevated, with a predominance of polymorphonuclears. It may reach as high as 20,000, but this is unusual. Sulfonamide treatment is of no avail. Therapy is entirely symptomatic and must be directed at the distressing cough, and in the severe cases at dyspnea. Complications are few. Sinusitis and bronchiectasis have been encountered. There were 3 fatal cases in a series of 122 in which the disease was definitely diagnosed. Two of the fatal cases were autopsied. They showed patchy areas of bronchopneumonia with purulent bronchitis. The alveoli contained much amorphous pink staining material and large macrophages filled with pigment. No inclusion bodies were found.—*Primary Atypical Pneumonia of Unknown Etiology, S. Gundersen, New England J. Med., November 23, 1944, 231: 697.*—(*H. Marcus*)

Atypical Pneumonia.—Roentgenographic findings in 178 cases of primary atypical pneumonia of unknown etiology are described. Eighty per cent of the lesions were in one of the lower lobes. As a rule, the infiltration is peribronchial; in cases with apparently lobar involvement lateral roentgenograms usually reveal a segmental, confluent bronchopneumonia. In one group of cases the pneumonia manifests itself as a homogeneous, usually slight density, in another the densities are flocculent; occasionally these two types may be coexistent. Of the cases observed 46.4 per cent belonged to the first group, 39.7 per cent to the second and 13.9 per cent to the third. In all three groups the appearance of infiltrations is preceded by a structural accentuation localized to the involved part of the lung. In the homogeneous group beginning resolution is indicated by a decrease in density, giving the lesion a moth-eaten appearance. Pleuritis with effusion is uncommon; lateral views may be necessary to differentiate it from parenchymal infiltrations. Elevation of a hemidiaphragm, interpreted as evidence of atelectasis, was observed in 38 cases. Occasionally Fleischner's lines are present, but other signs of atelectasis are usually absent. In the majority of cases anatomical resolution of infiltrations takes approximately two weeks. Nonresolution was seen in 5 patients, 3 of whom showed clinical, and 2 of the latter roentgenological, evidence of bronchiectasis. The 2 remaining cases had clinical signs of asthma.—*The Roentgenographic Manifestations of Atypical Pneumonia of Unknown Etiology, W. E. Crysler, Am. J. Roentgenol., March, 1944, 51: 280.*—(*P. Lowy*)

"Virus" Pneumonia.—Encouraged by the good results of roentgen therapy in the treatment of the severe cough that frequently persists long after the acute stage of "virus" pneumonia has subsided, the author employed radiation treatment in the early phase

of the disease. The following factors were used: 130–150 kv., 30 ma., 0.5 mm. Cu plus 1 mm. A 1 filtration, 50 cm. anode-skin distance, an average dose of 50 r (measured in air), 20 by 20 cm. portals. Fifty-six patients with clinical and roentgenological evidence of "virus" pneumonia were treated, and in 45 of them the results were good. Fever and cough gradually subsided, frequently within sixteen hours following treatment; roentgenological improvement did not run parallel to the clinical course. All patients (18) in whom treatment was instituted within five days of the onset of the disease responded favorably. Of 16 patients who had been ill for over two weeks before treatment was started, only 8 showed improvement. Roentgen therapy of "virus" pneumonia in the early stages of the disease is therefore recommended, with doses not exceeding 100 r.—*Roentgen Therapy of "Virus" Pneumonia, A. Oppenheimer, Am. J. Roentgenol., May, 1943, 49: 635.—(P. Lowy)*

Acute Pneumonitis.—During a four-month period 534 patients were admitted to a Naval Air Station Dispensary with the diagnosis of acute catarrhal fever; 145 of these patients had chest roentgenograms taken and 74 of these showed an area of pulmonary consolidation. The authors review some of the literature of acute pneumonitis (virus pneumonia, acute interstitial pneumonitis, atypical pneumonia or atypical bronchopneumonia of unknown etiology) and present their own findings. The disease is sometimes asymptomatic and is discovered accidentally. Other cases have but mild symptoms of an upper respiratory infection with moderately elevated temperature, and still others run high fever for several days; a second rise of temperature may also occur. A dry or slightly productive cough of varying severity is usually present. Physical signs over the chest may be entirely absent or inconclusive; sometimes, however, coarse râles persist after apparent recovery. The prognosis is generally good. Roentgenologically, the consolidation is most frequent in the lower and middle lobes, than in the hilar region, and is at least common in the upper lobes. At first a poorly defined hazy density is seen, which later becomes denser and then patchy. The area of consolidation is usually out of proportion to the paucity of clinical symptoms. The leucocyte count is normal or slightly elevated, occasionally diminished. The etiology is unknown; a virus is suspected by most investigators. At autopsy the principal findings are hemorrhage and edema. Mononuclear cells predominate in the exudate, fibrin is absent. Treatment is symptomatic; chemotherapy is ineffective and should be avoided. The authors present 7 illustrative cases and emphasize the frequency and communicability of the disease.—*Acute Pneumonitis, W. G. Scott & H. L. Jones, Jr., Am. J. Roentgenol., October, 1943, 50: 444.—(P. Lowy)*

Rib Fractures in Atypical Pneumonia.—Eighteen cases of fractured ribs were found in a review of the films of 500 consecutive cases of atypical pneumonia; 10 of the 18 were overlooked at the time of the original examination. The patients gave no history of trauma. The site of the fractures was in the anterior axillary or in the axillary line where the muscle fibres of the serratus anterior and externus obliquus abdominis exert opposing forces. It is generally believed that these fractures are due to violent coughing spasms which frequently occur in atypical pneumonia. The severity of the pneumonic process bears no relation to the occurrence of rib fractures. There was no evidence of calcium deficit, and all fractures healed promptly.—*Rib Fractures in Atypical Pneumonia, R. M. Harvey, Am. J. Roentgenol., November, 1944, 52: 487.—(P. Lowy)*

Ornithotic Pneumonia.—Three cases of atypical pneumonia apparently caused by the virus of psittacosis are presented, the literature is briefly reviewed, and the clinical, roentgenological and laboratory findings are described. After an incubation period of seven to fourteen days, signs of a generalized infection of varying severity appear, together with pulmonary involvement which in severe

cases may be wide-spread and associated with pleural effusion. Chest complaints are often disproportionately slight, and cyanosis more severe than one should expect from the roentgenographic findings. The white blood cell count is usually low or normal. The diagnosis of ornithotic infection can be substantiated by the finding of the virus during the acute phase or of complement fixing antibodies during convalescence; the titre of the latter must rise during the recovery period. Roentgenologically, the pulmonary lesions are similar to those seen in other cases of atypical pneumonia, that is, a central, patchy, migrating pneumonitis is most frequently demonstrated.—*Ornithotic Pneumonia, A. Melamed & J. M. Fine, Am. J. Roentgenol., May, 1944, 51: 548.*—(*P. Lowy*)

Pneumonitis with Malaria.—One hundred and twenty-five consecutive patients with pneumonitis associated with malaria, admitted to Gorgas Hospital in a period from January, 1942 through May, 1943, were studied. The most important group, comprising 70 per cent of the total, consisted of young white men belonging to the military personnel. In this group the incidence of pneumonitis associated with malaria as compared with that of uncomplicated malaria was 3.7 per cent. The disease occurred mainly during the "rainy" season and corresponded in general to the peak of the incidence of malaria and also of pneumonitis. Physical signs were absent in 36 per cent of the cases. Roentgen examinations revealed a high percentage of lesions in the lower lobes and of the lobular type. Estivo-autumnal malaria and tertian malaria were associated with pneumonitis to approximately the same extent. Negative results of cultures of sputum were reported in the majority of cases in which specimens were submitted for study. The leucocyte count was usually normal or low. Chemotherapy and antimalarial treatment were employed. There was only one death, which was due to cerebral malaria. The period of hospitalization was 5 to 70 days. Pneumonitis in malaria may be classified as follows: (*1*) atypical (possibly virus) pneumonitis, with inadequate response to therapy; (*2*) bacterial pneumonitis, with satisfactory response to sulfonamide compounds; (*3*) malarial pneumonitis, with favorable response to antimalarial therapy.—*Pneumonitis Associated with Malaria, I. L. Applebaum & J. Shrager, Arch. Int. Med., September, 1944, 74: 155.*—(*G. C. Leiner*)

Right Middle Lobe Pneumonia.—In reporting the case record of a patient with an acute pneumonia of the right middle lobe, the author takes occasion to point out the value of roentgenograms taken in the lateral projection for demonstrating the location of the right middle lobe and the fissures of the lung. —*La radiología del pulmón derecho y especialmente del lóbulo medio y cisuras, A propósito de una neumopatia aguda, M. M. del Carril, Rev. méd. d. Hosp. Ital. d. La Plata, June, 1944, 1:* 35.—(*R. Kegel*)

Fleeting Pulmonary Infiltrate.—A round, fairly dense, sharply bordered infraclavicular infiltrate with no peripheral reaction is described as occurring in a 31 year old asthmatic. X-ray examination twenty days later failed to show it. The author believes it was a small atelectatic focus, provoked by a localized bronchial spasm or mucosal congestion. The differential diagnosis lies between tuberculous round infiltrate, hydatid cyst, primary or metastatic tumor and the above which is uncommon.—*Infiltrado redondo fugaz, M. Fierro Vignoli, Hoja tisiol., June, 1944, 4: 89.*—(*J. S. Peterson*)

Fleeting Pulmonary Infiltrates.—The case histories of 2 patients with transient pulmonary infiltrates are reported. In the first patient an infiltration in the right lower lobe was followed by an infiltration in the left midfield. The second patient had only one infiltration in the left midfield. In both cases the eosinophils were 4 per cent and in the feces of the first patient parasites were found. In both cases the infiltrates disappeared entirely within ten days. The catarrhal upper respiratory symptoms which had been present

at the onset of this condition in both patients make it appear likely that the pulmonary infiltrations were part of an influenzal disease. (Bronchopneumonia and tuberculosis were excluded on the basis of the X-ray appearance and the clinical course.) It is assumed that previous diseases (tuberculosis in the first and Basedow's disease in the second case) had caused a vascular instability in the lungs which became clinically manifest under the influence of the influenza virus.—*Contributo allo studio degli infiltrati polmonari fugaci, M. Mattina, Lotta contro la tuberc., 1941, 12: 707.—(G. Simmons)*

Fleeting Pulmonary Infiltrates.—Transient pulmonary infiltrates are classified into three different categories: (*1*) transient infiltrates occurring in individuals who have tuberculosis in other pulmonary districts. These processes are considered to be tuberculous epiphenomena on a vascular basis. Their resolution is relatively slow and usually takes several months; (*2*) transient pulmonary infiltrates associated with acute forms of different pulmonary conditions, especially pneumonia and bronchopneumonia; (*3*) transient pulmonary infiltrates with eosinophilia as first described by Loeffler and believed to be on an allergic basis.—*Les ombres radiologiques pulmonaires fugaces. Essai de classification et d'interprétation à propos de quelques observations inédites, E. Delbecq & A. Garnier, Arch. méd.-chir. appar. respirat., 1942, 15: 17.—(G. Simmons)*

Loeffler's Syndrome.—Few if any cases of Loeffler's syndrome have come to autopsy so far, because the condition is benign and never is fatal. In this paper 4 cases of transient eosinophil pulmonary infiltrate are reported. The patients died of another cause. Macroscopically these infiltrates were not different from any bronchopneumonic focus. Microscopically the great number of eosinophil cells was striking; otherwise these infiltrates showed all the characteristics of an inflammatory process. Charcot-Leyden's crystals were found in only one case. Interesting is the presence of concomitant eosinophil infiltrates in other organs. The problem of the origin of this condition is not yet clear, but animal parasites appear to be the main cause, especially the ascaris. Loeffler's pulmonary infiltrate is considered to be an inflammatory allergic reaction toward different antigens which can arrive in the lungs by different ways.—*Das eosinophile Lungeninfiltrat. Pathologische Anatomie und Pathogenese, H. v. Meyenburg, Schweiz. med. Wchnschr., 1942, 11: 809.—(G. Simmons)*

Eosinophilic Lung Infiltrations.—A soldier of 23 was taken ill with protean symptoms of respiratory disease. Examination of the peripheral blood showed leucocytosis from 16,000 to 41,000 with up to 80 per cent eosinophils. The chest X-ray film showed bilateral pulmonary infiltrations which were fleeting in character and repeatedly resolved and reappeared. Thorough work-up failed to reveal any etiological agent, although a few ova of *Trichuris trichiura* were found in 2 out of 32 stool examinations. Sputum and urine examinations were negative for parasites or pathogenic bacteria. Intracutaneous tests for allergens, including tests for trichinella, were negative. Blood agglutination studies for brucellosis were negative and no filariae were found on examination of midnight blood smears. Sternal puncture showed a predominance of eosinophils. The patient was cured after an illness of nearly three months by the injection of biweekly doses of 0.06 g. of Mapharsen. Ten such treatments were given. This therapy was employed following a report by Weingarten from India who cured similar cases with arsenicals.—*Transitory Lung Infiltrations Accompanied by Eosinophilia, H. Miller, New England J. Med., January 4, 1945, 232: 7.—(H. Marcus)*

Lung Abscess.—From the point of view of etiology, pathogenesis, prognosis and treatment, several types of lung abscess must be distinguished. The most common abscess is the solitary putrid abscess. It is caused by anaerobic organisms, notably fusiform bacilli, spirochetes, streptococci and vibriones, and it

occurs most frequently when the natural defensive mechanisms guarding the lung from infection are in abeyance, namely, during anesthesia, unconsciousness and sleep. Infected material lodges in a terminal bronchiole and there sets up a localized area of gangrene. This area enlarges until the local defense mechanisms are sufficient to wall it off, usually before the pleura is reached. After seven to ten days the contents of the infected area liquefy partially and necrotic lung slough and pus are evacuated through the bronchus. When evacuation is complete, the abscess heals spontaneously. More often only partial evacuation takes place and surgery is needed to effect a cure. Putrid abscesses following upon abdominal operations usually carry a 60 per cent mortality and it is better to leave them alone. Other putrid abscesses have a good chance for permanent cure by surgery. Aerobic abscess of the lung is the result of a diffuse bronchopneumonia. Several lesions coalesce, and usually several small abscesses are formed. The smaller ones tend to heal spontaneously upon discharging their purulent contents. The large ones may need surgical drainage, although the cures from surgery are not as dramatic in this type as in the anaerobic type. The prognosis is good. Staphylococcus abscesses occupy a special place. Multiple abscesses may occur during the course of a generalized septicemia. The prognosis is good if the patient survives his infection. Another type follows upon a staphylococcus pneumonia. Multiple lung abscesses form and the patient is extremely ill. The prognosis is grave. Occasionally hematomata following injury to the chest break down and suppurate. This is unusual, because resorption of the extravasated blood is the rule. However, when the hematoma becomes infected an abscess may form which demands surgical treatment. Bronchial abscesses are occasionally seen. They occur when the bronchus becomes occluded by a foreign body of mineral matter, such as a tooth. Collection of pus and exudate behind the obstruction forms the abscess but its location is entirely intrabronchial and the parenchyma remains uninvolved.—*Lung Abscess, N. R. Barret' Lancet, November 18, 1944, 247: 647.*—(*H. Marcus*)

Suppurative Bronchopneumonia.—Suppurative bronchopneumonia is not rare. In a period of more than ten years 120 cases have been studied at Mount Sinai Hospital. The incidence of this disease shows seasonal variations similar to other forms of bronchopneumonia. It occurs in all age groups, however one-third of the patients were children under the age of 10 years. The history was usually that of an infection of the upper respiratory tract in previously healthy persons. The pathological features consist of a severe bronchopneumonia involving one, several or many portions of the lungs. Spread from primary areas appears to be chiefly by the mechanism of spillover. Varying degrees of suppuration and necrosis occur within the affected segments of lung, usually with the formation of single or multiple foci of liquefaction within areas of bronchopneumonia. If spectacular necrosis is present, the term of "necrosuppurative bronchopneumonia" is applied. Suppurative pleuritis is a frequent complication. Complete recovery or death in the acute stage is common. A chronic phase with formation of abscess or chronic interstitial pneumonia and bronchiectasis is rare. Bacteriologic examination was done from pus obtained by aspiration or at operation, not from sputum. Most freqeuntly found were *Staphylococcus aureus*, hemolytic streptococci, pneumococci, *Streptococcus viridans*. The roentgen evidence of one or more areas of rarefaction in the midst of pneumonic infiltration was found to be pathognomonic. Tomography often only revealed the presence of areas of rarefaction. Symptoms were fever, cough, expectoration of purulent sputum, usually in large amounts, pleuritic pain. The signs are similar to those of other bronchopneumonic lesions. The following classification of the disease is given: I. Basic forms: *A*. Localized suppurative bronchopneumonia. *B*. Diffuse suppurative bronchopneumonia. II. Secondary suppurative bronchopneumonia: *A*. Postoperative. *B*. In cachexia. *C*.

From other predisposing causes. III. Surgical forms: *A.* Pulmonary abscess. *B.* Empyema and pyopneumothorax; (*1*) With proved pulmonary focus. (*2*) Without proved pulmonary focus. IV. Complications of suppurative bronchopneumonia: *A.* General: (*1*) Cerebral. (*2*) Septic. *B.* Local: (*1*) Lattice lung. (*2*) Bronchiectasis. (*3*) Pericarditis. (*4*) Mediastinitis. The disease is self-limited and essentially unaffected by chemotherapy or serum therapy. Surgical complications within the thorax are common. Pulmonary abscess is an unusual complication. Of these 120 cases, 29 died (24.2 per cent), mostly due to intrathoracic complications.—*Acute Suppurative Bronchopneumonia, H. Neuhof & A. Thomas, Arch. Int. Med., January, 1945, 75: 45.*—(*G. C. Leiner*)

Bronchiectasis.—At a Station Hospital, bronchiectasis was found to be the most frequent chronic pulmonary condition. Ninety-five patients, most of whom were admitted with acute respiratory infections, were observed for several weeks and months, and an attempt was made to correlate the prebronchographic roentgenological findings with the results of bronchography. Frank bronchiectasis was demonstrated in 37 cases, minimal or questionable bronchiectasis in 24 and no bronchiectasis in 34. Slowly resolving basal bronchopneumonia was the most frequent prebronchographic abnormality. This was found in 53 of the 95 cases observed, and in 28 of the 37 in whom later frank bronchiectasis was revealed by bronchography. Prominent pulmonary markings are even more frequently seen, but are less reliable in indicating bronchiectasis. Six of the 37 frankly bronchiectatic cases had recurrent bronchopneumonia in the same area of the lung. The finding of a shrunken contracted area of the lung also serves to raise the question of bronchiectasis; complete segmental atelectasis was not observed in this series. Although bronchiectasis may be strongly suspected from plain roentgenograms a definite diagnosis cannot be established without bronchography.—*The Diagnosis of Bronchiectasis in Young Adults, Prebronchographic Roentgen Manifestations Observed among Military Personnel, W. A. Evans & L. J. Galinsky, Am. J. Roentgenol., May, 1944, 51: 537.*—(*P. Lowy*)

Bronchiectasis.—The incidence, pathology, pathogenesis, bacteriology, symptomatology, course, importance and treatment of bronchiectasis are discussed in detail, in order to arrive at some means of prevention. Modern diagnostic methods reveal that bronchiectasis is not at all uncommon. Among adults, males are somewhat more frequently affected, while in children the sex ratio is about equal. The disease occurs in all age groups, the onset being before the age of 10 in half the cases; the diagnosis is most frequently made in the third decade. The clinical entity of bronchiectasis requires the presence of dilated bronchi and an associated infection; the latter is absent in so-called dry bronchiectasis. Dilatation of the bronchi is in itself the result of an infection which destroys the bronchial wall and thus produces cylindrical, fusiform or saccular bronchiectasis. If the bronchial wall is perforated in the course of the infectious process abscess cavities are formed. Bronchiectasis does not tend to spread beyond the original site of involvement; however, in the involved area the changes are progressive. The left lower lobe is most frequently affected, perhaps because of the anatomical characteristics of the left bronchial tree. It is now held probable that bronchial obstruction is essential in the pathogenesis of bronchiectasis. When inspissated secretion, tumor, granulation tissue or scarring occludes a bronchus collapse of pulmonary tissue distal to the obstruction occurs, with resulting overaeration of the surrounding lung tissue and disturbance in the normal pressure conditions. Traction is thus exerted on the weakened bronchi. In partial bronchial obstruction with ball-valve effect, the inspiratory increase in pressure may be sufficient to dilate pathological bronchi. From the etiological point of view bronchiectasis may be congenital or acquired. Among the latter chiefly infectious, chiefly obstructive and miscellaneous groups can be differentiated,

with several subdivisions in each group. There is no single organism responsible for the development of bronchiectasis. The salient clinical features are cough, expectoration and occasional hemoptysis. In addition, bouts of fever, dyspnea, night sweats, anemia and loss of weight may be present. The cough is usually worse in the morning. The sputum is frequently foul and "chunky," its amount varies. Hemoptysis is common in bronchiectasis and is due to the presence of vascular granulation tissue. Failure to eliminate bronchial secretions produces fever, the degree of which depends upon the amount retained. On physical examination there are not pathognomonic signs of bronchiectasis. Clubbing of the fingers is frequently found. The clinical symptoms and signs and the routine roentgenograms are not sufficient for the diagnosis. Bronchoscopy and bronchography are essential. The former may reveal congenital anomalies, foreign bodies or signs suggestive of bronchiectasis, although the dilated bronchi themselves usually cannot be reached with the bronchoscope. Bronchography, on the other hand, may unquestionably demonstrate the presence of bronchiectasis. Although its onset is usually during childhood, in the majority of cases, bronchiectasis does not become clinically manifest until early adult life. The patients are subject to frequent protracted "colds," and eventually toxic symptoms (lassitude, loss of weight, etc.) appear and cough and expectoration persist even between the acute flare-ups. Repeated attacks of pneumonia are apt to occur and one of these may be fatal. The mortality rate of bronchiectasis as reported in the literature varies between 23 and 38 per cent. Medical treatment consists of postural drainage and general and symptomatic measures. Bronchoscopic drainage affords but temporary relief. The value of radiotherapy in converting an infected bronchiectasis into a dry one is doubtful. Lobectomy and pneumonectomy promise to give permanent cure in cases limited to one lobe or one lung. Surgical treatment should be resorted to if the disease shows definite signs of progression, and then the operation should be performed as early as possible, before the patient's general condition declines. More than half the cases are unilateral and thus suitable for surgical treatment. In the prevention, diagnosis and thorough treatment of infection, apt to produce bronchiectasis, are important. Such infections include the common cold, influenza, measles, whooping cough and pneumonia. Cases of pneumonia should be followed roentgenologically until complete resolution, and if this is delayed, bronchoscopic and bronchographic examination should be performed. Bronchial obstruction of whatever cause (foreign bodies, broncholiths, thick bronchial secretions, strictures, tumors) should be promptly investigated and if possible relieved. In general, roentgenography and bronchoscopy are the most important diagnostic procedures in the prevention of bronchiectasis. The proper use of these and the education of the medical profession and of the lay public are required if the incidence of bronchiectasis is to be reduced.—*A Plea for the Prevention of Bronchiectasis, K. Kornblum, Am. J. Roentgenol., March, 1944, 51: 202.—(P. Lowy)*

Irradiation Pneumonitis.—A case of Hodgkin's disease treated by X-ray is described. The patient, a 52 year old white male, presented tender enlarged nodes in the supraclavicular fossae and in the left axilla. A biopsy specimen from a node showed typical Hodgkin's disease on microscopic examination. A roentgenogram of the chest showed moderate widening of the mediastinum, due to enlargement of the lymph node and slight infiltration extending into the lung tissues adjacent to the hila, which was more marked on the left side. Over a period of 9 months the patient received many X-ray treatments. At first the lung fields were shielded, but later this was discontinued. Near the end of the course the mediastinum was intensively irradiated, 3,836 r being administered over a period of 17 days. A month later he became very short of breath, with rapid respirations and slight cyanosis of the face and lips. Roentgenograms of the chest showed infiltration of

the lower two-thirds of each lung becoming increasingly dense towards the base. He was then given deep therapy to the posterior mediastinum and to the lumbar regions. Death occurred shortly, apparently from asphyxia. It is to be noted that the patient developed marked respiratory symptoms shortly after a course of irradiation to the mediastinum where the adjacent lung tissues necessarily were not shielded. Roentgenograms at this time showed an extensive pneumonitis. At autopsy the lungs exhibited lesions similar to those produced experimentally by over-irradiation. A patchy exudate composed of edema fluid, a few red and white cells, fibrin and fibrinous-hyaline membrane was found involving the greater portion of each lung. There was wide-spread organization of this exudate. A few minute microscopic foci of Hodgkin's disease were found in the bronchial lymph nodes and in the pericardium. No Hodgkin's tissue was found in the lungs. Death occurred from excessive irradiation. (Illustrated).—*Irradiation Pneumonitis: Report of a Case, T. O. Alexander, Bull. Johns Hopkins Hosp., October, 1944, 75: 199.*—(*J. S. Woolley*)

Hydatid Cyst.—The authors report an instance of an hydatid cyst of the spleen rupturing into a bronchus. The patient was a 32 year old married woman who entered the hospital complaining of malaise, anorexia, chills, fever, left pleuritic pain, cough and dyspnea of 40 days' duration and of abundant mucopurulent expectoration of several days. The positive physical findings were signs of a pleural effusion at the left base, an enlarged spleen, and an umbilical hernia. There was an eosinophilia of 6 per cent. Ghedini's reaction was doubtful and Cassoni's negative. There were no tubercle bacilli in the expectoration but the hooklets of taenia echinococcus were found. An X-ray film of the chest showed a marked elevation of the left leaf of the diaphragm. Laparotomy disclosed the complete destruction of the spleen by a hydatid cyst. After formol injection the cyst was marsupialized. The postoperative course was satisfactory. Three years later the patient returned for repair of the umbilical hernia. An X-ray film of the chest showed no abnormalities. There was no eosinophilia. Both Ghedini's and Cassoni's reactions were negative. Laparotomy revealed absence of the spleen and scar formation between the tail of the pancreas and the diaphragm. There was no evidence of cyst formation in the abdominal cavity. Hydatid disease of the spleen is infrequent (3 per cent). Morel's 20 cases comprised 17 cysts primary in the spleen and 3 secondary to rupture of an abdominal cyst. Perisplenic adhesions are common especially with the diaphragm. Symptoms usually do not appear until the cyst is large.—*Quiste hidático del bazo abierto en bronquios, D. Unchalo, J. M. Mainetti & C. Cuculicchio, Rev. méd. d. Hosp. Ital. d. La Plata, June, 1944, 1: 173.*—(*R. Kegel*)

Bronchogenic Cysts.—The differential diagnosis between true, bronchogenic cysts and acquired lesions is sometimes impossible. The former are probably congenital and are usually situated in the mediastinum, the latter may represent the end-results of lung abscess or bronchiectasis. Various tumors of the lung and mediastinum also enter into the differential diagnosis. Fifteen cases of proved bronchogenic cyst are described. In some of them the histological picture was not quite typical of, but very similar to, that of bronchogenic cysts; in others no histological examination was done but the gross appearance of the lesion made the diagnosis practically certain. Three additional cases are presented in which the diagnosis was questionable. Roentgenologically, bronchogenic cysts appear as smooth, round or ovoid shadows which sometimes show change in shape with change in the patient's position. The demonstration of tracheal attachment of the mass and of intramural, extramucosal involvement of the esophagus corroborates the diagnosis. The cyst may or may not show a fluid level; if it does, the differentiation from lung abscess may be impossible. Clinical symptoms, if there are any, also are suggestive of lung abscess.—

The Roentgenologic Appearance of "Bronchiogenic" Cysts, L. L. Robbins, Am. J. Roentgenol., September, 1943, 50: 321.—(P. Lowy)

Cystic Disease and Dextroaortic Arch.—A 40 year old male teacher is reported, who developed a febrile acute pulmonary congestion at the right base with diffuse bronchitis. The upper zones of both hemithoraces were hyperresonant and there was no indication of pulmonary activity, contrasting with a rich clinical phenomenology in the lower zones. The patient recovered several days later. A serial radiological study revealed in the left upper and middle pulmonary zones a giant bulla of subpleural emphysema. In the right lung there was a giant air cyst and a congestive zone. There were scattered annular shadows. The aortic arch shadow was atypical, being to the right and at this level the trachea was displaced forward showing also a sinistroposition, pathognomonic of aortic dextroposition. There was coexistence of a double congenital malformation, the dextroposition of the aortic arch and the cystic disease, the former unquestionably being of embryonal origin and the latter was also considered as congenital due to: (*1*) it's coexistence with another congenital malformation, (*2*) the air cyst shadows remaining unchanged after two and one-half years of observation, (*3*) the coexistence of some other annular shadows in the neighborhood of the giant air cysts that also remained unchanged, and (*4*) the absence of pulmonary diseases during the past life of the patient. In this case there was also coexistence of the two cystic types of congenital pulmonary alterations, namely, the air cyst and the bulla of subpleural emphysema.—*Disgenesia pulmonar, bilateral, de forma quistica gigante y dextroposición del cayado aórtico, J. Queirel & M. Moreau, Rev. Asoc. méd. argent., January–February, 1944, 58: 14.—(J. Badell)*

Annular Shadows.—Five cases are reported in which annular shadows of uncommon type were encountered. In all the cases the onset of the disease was with evidence of infection, at first localized (sore throat, mastoiditis, arthritis, etc.), then systemic; blood cultures were positive (for *Streptococcus hemolyticus* and *Staphylococcus aureus*, respectively) in 2 patients. Within a few days multiple rounded shadows appeared in both lung fields. Soon central rarefaction occurred, resulting in thick walled annular shadows containing air and fluid. All patients were given one or several of the sulfonamides. Four patients recovered; one of these developed areas of localized empyema which were subsequently drained, while in the 3 others several months after the acute illness the annular shadows were still visible, but by this time closely resembled emphysematous blebs. One patient died and was autopsied. On the basis of the postmortem findings, it is suggested that the origin of the annular shadows may be septic thrombosis of the bronchial artery, with resulting infarction and breakdown.—*Annular Shadows of Unusual Type Associated with Acute Pulmonary Infection, L. R. Sante & C. E. Hufford, Am. J. Roentgenol., December, 1943, 50: 719.—(P. Lowy)*

Nontuberculous Cavitation.—Although the most frequent cause of pulmonary cavitation in the upper lobes is tuberculosis, the finding of 1.5 per cent nontuberculous cavities in an autopsy material of 2,000 cases with cavitation warns that causes other than tuberculosis have to be considered in the diagnosis. Eight cases are presented, all of whom had cavitation but no tuberculosis. The 8 diagnoses were the following: anthracosilicosis, cystic disease of the lung, actinomycosis, aortic aneurysm causing pulmonary necrosis, bronchogenic carcinoma with parenchymal necrosis, lung abscess, infection with Friedländer's bacillus, bronchiectasis. In the majority of these cases tuberculosis was diagnosed at first, although only one of them had a single positive sputum.—*Non-tuberculous Pulmonary Cavitation, L. Nathanson & P. Morgenstern, Am. J. Roentgenol., January, 1944, 51: 44.—(P. Lowy)*

Interstitial Emphysema of Lungs.—There are many clinical cases in which the presence

of air in the tissues of the lung is not suspected, and in which its effects have been regarded as being produced by the primary disease itself, rather than by the interstitial air. There are many other cases in which air is recognized as being present in the pleural cavities, in the subcutaneous tissues of the neck, trunk, etc., in the retroperitoneal spaces or in the peritoneal cavity, but in which the method of escape of air and the route it followed is misunderstood. Because this air in the pulmonary interstitial tissues may cause death through airblock, if it is not removed, the clinician must become aware of the conditions which predispose to its appearance. These conditions may be divided into three categories: (*1*) those in which there is first an atelectasis of some part of the lung, followed by hyperinflation in adjoining regions of the same lung or in the opposite lung; (*2*) those in which there is a general overinflation with or without increased intraälveolar pressure; (*3*) those in which there is evident a decreased blood supply to the pulmonary vessels preferably either with increased intraälveolar pressure or with hyperinflation. The mode of escape of air is the same in these three classes, namely, through ruptured alveolar bases into the sheaths of the pulmonary vessels, and the clinical picture may be the same in all, with any of the following conditions either alone or in combination: air in the mediastinum; in the thoracic cavity; in the subcutaneous tissues of the face, neck, chest, axillae and body; in the retroperitoneal spaces from whence it may rupture into the peritoneal cavity; around the pericardium, in which event a pericardial knock is heard. There is dyspnea and cyanosis when the pressure in the mediastinum rises too high, limitation of respiratory movements with the chest becoming fixed in a position of maximal or submaximal inspiration when the air either distends the mediastinum or gets into the connective tissue septa of the lung. The air may gradually be resorbed and the patient recover, or it may increase in severity and the patient may die. The precipitating cause of this train of events may occur in a wide variety of conditions, but is always a pressure gradient from air in the alveoli to perivascular sheath or underlying septa, leading to alveolar rupture and formation of pulmonary interstitial emphysema. Air leaks from the overstretched alveoli into the sheaths or adventitia of small branches of the pulmonary arteries and veins (factor A); or from alveoli, surrounding blood vessels not filled to the normal extent with blood (factor B), the alveoli being either normally expanded or hyperinflated, under atmospheric pressure or pressures above atmospheric. The bubbles of air press upon the vessels occluding their lumina producing airblock, and may leak into the interlobular connective tissue, causing airblock. It makes its way along the vessel sheaths to the mediastinum where it presses upon the large vessels at the base of the heart. It may be removed from the anterior mediastinum. It may make its way upwards into the neck, face and axillae, thence down over the chest and arms, giving rise to subcutaneous (erroneously called "surgical") emphysema. It may make its way downwards along the aorta and esophagus into the retroperitoneum and may rupture into the peritoneal cavity. Symptoms produced by air in the abdominal cavity may simulate acute abdominal conditions for which operation may be mistakenly performed. It may make its way forward over the heart, whence it may give rise to a loud crunching sound, "Hamman's sign," with each heart beat. It may travel laterally into the vessel sheaths of the other lung, or backward along sheaths of the same lung into areas in which there is no leakage. It may rupture the mediastinal wall producing pneumothorax. Collapse of the lung tends to stop the leak, except in cases in which there is violent cough. Tension pneumothorax may result from air escaping from the mediastinum, but the tension is built up not during inspiration, but during forced expiration of cough or when the glottis is closed and the intrapulmonary pressure rises above atmospheric. Air continues to leak as long as the factor initiating the original break is operative. In some instances the leak appears to be favored merely by respiratory movements, especially

if they are of a dyspneic character. The factor responsible for moving the air along the sheaths is the lengthening and shortening of the bronchi in normal respiration. Pain may possibly be caused by air pressing upon the pulmonary and mediastinal vessels, simulating angina pectoris. Circulation is interfered with by the collapse of the pulmonary vessels producing airblock, causing venous stasis and giving rise to cyanosis. Respiration is interfered with by the splinting action of the air in the connective tissues of the lung causing airblock, preventing the escape of air in expiration and giving rise to dyspnea. The heart action may be interfered with in three ways: (*1*) by being pressed upon by the distended lungs which prevent its filling; (*2*) lack of blood to fill it because of systemic and pulmonic venous congestion, arising through pressure by air bubbles on vessels, and stasis; and (*3*) by direct pressure upon it by air bubbles in the precordium, and in the posterior mediastinum. Factors predisposing to leakage are apparently toxins of certain infectious diseases, particularly influenza; and perhaps an inherited constitutional weakness of the alveolar walls. Once the leakage has begun, the pressure necessary to continue the leak need not be so high as that initiating the rupture. When air escapes from the mediastinum into the subcutaneous tissues, or into the retroperitoneum or even into the pleural cavities, provided that it does not produce bilateral pneumothorax, or a tension pneumothorax, the condition is likely to be benign, since the pressure in the mediastinum is relieved. If the leak continues and builds up higher pressures in the mediastinum than can be relieved by the avenues of escape, the condition, originally benign, may become malignant. It is when the air cannot escape from the mediastinum and the pressure rises too high that the condition becomes malignant. Air in the mediastinum and interstitial tissues of the lung may be occult, unrecognized by means of visible manifestations. It accompanies a wide variety of clinical conditions and respiratory diseases. Especially when it is occult it may be malignant; when it is malignant it can be fatal. When its presence is diagnosed, it may be withdrawn and thus the patient's life may be saved. (Authors' summary.)—*Malignant Interstitial Emphysema of the Lungs and Mediastinum as an Important Occult Complication in Many Respiratory Diseases and other Conditions: An Interpretation of the Clinical Literature in the Light of Laboratory Experiment, Madge Thorlow Macklin & C. C. Macklin, Medicine, December, 1944, 23: 281.*—(*G. C. Leiner*)

Emphysema of Lung.—Senile or postural emphysema is usually not sufficiently serious to warrant special therapy. Extreme degrees of respiratory dysfunction are seen in obstructive or so-called chronic hypertrophic emphysema. This type is seen in the young or middle aged, and there is usually a long history of chronic respiratory infections, such as bronchitis, asthma and repeated attacks of pneumonia. Diminution in function is often so extreme that patients are dyspneic even at rest. The vital capacity may not exceed 1,000 cc. and, even where the actual measurement of vital capacity appears good, there may still be extreme disability due to abnormal prolongation of the expiratory phase, so that the patient cannot utilize his capacity. Although it may not be apparent on physical examination, a spastic element is frequently present, and the therapeutic test with inhalation of nebulized adrenalin or injection of adrenalin in oil is always worth while. Oxygen therapy is of extreme usefulness in raising the oxygen saturation of blood and thus correcting the chronic state of anoxia in which these patients live. The arterial blood in a bad case may have an oxygen saturation of not more than 60 per cent. Discontinous oxygen therapy at home is entirely practicable. It provides the patient with the much needed rest and brings about an improvement in the patient's general condition so that the amount of oxygen can be gradually reduced. In some cases abdominal belts may be useful.—*The Abnormal Physiology of Chronic Pulmonary Emphysema, A. F. Goggio, New England J.*

Med., November 16, 1944, 231: 672.—(H. Marcus)

Emphysematous Bullae.—A case of lung abscess in a 15 year old female is described. Although on two occasions acid-fast bacilli were found in the sputum on concentration, lung abscess was diagnosed and surgical drainage was performed. On careful examination, including guinea pig inoculation, no evidence of tuberculosis was found in the pus evacuated from the abscess or in the abscess wall. The patient steadily improved and was discharged ten months after operation. Shortly before discharge, planigrams revealed several thin-walled annular shadows at the site of the previous abscess. These shadows are believed to represent emphysematous bullae, due either to a check-valve mechanism, or to the surgical interference, or to both.—*Postoperative Emphysematous Bullae following Lung Abscess, W. R. Oechsli, Am. J. Roentgenol., August, 1944, 52: 145.—(P. Lowy)*

Emphysema of Mediastinum.—The diagnosis of mediastinal emphysema is difficult. The case presented was a man, 24 years of age, who in 1940 noticed a sharp pain in his left arm and left axillary region. This pain increased with all movements. He had intense palpitations of the heart and at this time a loud pericardial friction rub was heard. The patient was X-rayed and an electrocardiogram was taken, but no evidence of any disease, pulmonary or other, could be found. Four years later he reëntered the hospital complaining of a sharp pain in the lower left chest radiating into the left shoulder, of anxiety and palpitations. Three days after the onset a friction rub appeared in the precordial region and a high-pitched râle in the left axillary region. Neither fluoroscopy nor X-ray films revealed any abnormalities. The rubbing sound over the pericardium is explained by the movements of the heart. The compression and decompression of the air present in the mediastinum causes the pulse-like rhythm. The absence of radiological signs is explained by the small quantity of air which entered the mediastinum. The patient made a complete recovery.—*Un nuevo caso de enfisema espontaneo del mediastino (forma frustra), E. G. Fongi, Prensa méd. argent., November 22, 1944, 31: 2380.—(W. Swienty)*

Spontaneous Pneumothorax.—Seven soldiers with spontaneous pneumothorax were admitted to the Station Hospital at Seymour Johnson Field in a period of 12 months. The patients were at an age of 19 to 23 years. The attack was preceded by exercise in 3 cases only; it occurred during sleep in one case. The main symptom was chest pain. Physical findings were present only when the lung was collapsed more than 50 per cent. The time required for reëxpansion of the lung was 3 to 37 days. None of the patients had pulmonary tuberculosis. It is believed that spontaneous pneumothorax was produced by the rupture of subpleural emphysematous blebs. It is stressed that "recurrent bouts of chest pain, in otherwise normal, young individuals, unaccompanied by friction rub or fever, but often associated with fine crepitant râles in the area of pain, suggest spontaneous pneumothorax and warrant X-ray study."—*Spontaneous Pneumothorax in Soldiers, P. P. Pease, L. G. Steuer & A. S. Chapman, Bull. U. S. Army Med. Dept., November, 1944, No. 82: 102.—(G. C. Leiner)*

Spontaneous Hemopneumothorax.—A case is reported of a 28 year old white woman who had a copious hemoptysis and, as a therapeutic measure, a pneumothorax was induced. Collapse therapy was continued among other reasons because of positive sputum. Pneumothorax was maintained by refills performed every 12 or 14 days. The only existing adhesion was severed. The patient awakened suddenly 18 months later with an intense pain in the right lower chest accompanied with dyspnea. She had to be hospitalized, presenting all the symptoms of extreme illness, pallor, coldness, cyanosis, weak pulse, tachycardia, blood pressure 90/70. As the patient had had amenorrhea for two months a gynecological examination was done to rule out a rupture

of an extra-uterine pregnancy, but probable signs of a normal two and one-half month pregnancy were found and confirmed by the Friedman test. A therapeutic abortion was performed later by Boero's procedure. There was evidence of the existence of a collection of fluid in the right pleural cavity with deviation of the mediastinum and its organs to the opposite side and the existence of a shock syndrome with all the characteristics of an internal hemorrhage. It was confirmed by radiological examination; and an exploratory thoracocentesis revealed that there was a collection of pure blood. The intrapleural pressure was 28. The patient was very ill for the first days and there were periods of true shock with unconsciousness and marked hypothermia; the systolic pressure was 70 with no appreciable difference with the diastolic; tachypnea and cyanosis. All symptoms began to disappear after 4 or 5 days. Treatment consisted of evacuation of the pleural cavity and blood transfusion. The most likely cause of the hemothorax was the rupture of a subpleural "bulla" or "bleb". In the presence of a spontaneous hemopneumothorax there is (*1*) sudden diminution of breathing parenchyma due to pulmonary collapse, (*2*) decreasing of circulating blood due to internal hemorrhage and (*3*) decreasing of the functional capacity of the heart due to decreased amount of circulating blood, to shifting of mediastinum and to compression of right auricle.—*Hemoneumatórax espontáneo agudo en el curso de un neumotórax en mantenimiento: Primera communicacion mundial, R. Ballestero, Rev. de tuberc. d. Cuba, October–December, 1943, 7: 503.*—(*J. Badell*)

Anthracosilicosis.—The case described is that of a 63 year old male who worked as a pick miner in a soft coal mine from 1884 to 1896. One month before death an X-ray film of the chest showed irregular nodulation in both lung fields with the exception of the apices, and a conglomerate density in each subclavicular region. The sputum was negative for tubercle bacilli on smear. A tuberculin test with 1:100 mg. of OT was negative. Five weeks after admission the patient died of carcinoma of the stomach. At autopsy 200 cc. of amber colored fluid was found in the right thoracic cavity. The surface of both lungs was black. There was a large cavity in the upper lobe of each lung; the cavities were filled with foul smelling tarry fluid. Many small, hard, black nodules were scattered throughout both lungs. Histological examination revealed conglomerate and discrete hyaline fibrosis with heavy coal pigmentation. No evidence of tuberculosis or metastatic carcinoma was found. The walls of the cavities contained free silica in sufficient amount to warrant the diagnosis of silicosis.—*Liquefaction Necrosis in Bilateral Symmetrical Conglomerate Lesions of Anthracosilicosis of the Lung: Report of Case, B. J. McCloskey, Am. J. Roentgenol., July, 1943, 50: 42.*—(*P. Lowy*)

Pregnancy after Lobectomy.—A considerable number of pregnancies following thoracoplasty are on record, but only one case of pregnancy after lobectomy has so far been reported. The author reports a woman in whom lobectomy was performed in 1937 for bronchiectasis of the left lower lobe. This patient was delivered of a normal child in January, 1943 and, although she was in labor for more than fifty hours, no cardiac or respiratory embarrassment was noted at any time. The patient was able to return home after sixteen days and nurse her child in addition to carrying on her household duties. Respiratory impairment after lobectomy is not due to removal of one or more lobes, but to persistent compensatory overdistention of the remaining lung. This is a variable factor and a successful lobectomy or pneumonectomy cannot be regarded as sufficient reason for therapeutic abortion.—*Pregnancy after Pulmonary Lobectomy, A. G. Bryce & E. M. Mills, Lancet, December 16, 1944, 247: 786.*—(*H. Marcus*)

Complications after Lobectomy.—The author describes the pulmonary complications after the removal of the inferior lobe. The important factor is a quick reëxpansion of the

superior lobe on the operated side which generally occurs on the third to fifth postoperative day. The movements of the diaphragm have to be normal and the pleural cavity should be free of liquid or air. A negative intrathoracic pressure must be maintained. It must be made sure that the remaining lobe is covered with elastic pleura and its bronchus patent. If it is adherent to the pleura it should be mobilized. Postoperative collapse of the superior lobe occurred almost exclusively in patients operated on for bronchiectasis. The author describes the preoperative preparation of the patient and emphasizes the necessity of a bronchoscopy and study with iodized oil six weeks before the operation, which should be done in spring or summer. As an anesthetic, he uses ether and oxygen. Individual section and ligature of the structures of the root of the lung, injection of the mucosa of the bronchus with 95 per cent carbolic acid and alcohol is done. The patient is kept for several days in an oxygen tent and is instructed to cough every hour on the first day in order to avoid accumulation of blood and mucus in the bronchus. He receives sulfadiazine, 0.03 to 0.04 g. per pound of body weight. Despite a good technique, the superior lobe does not reëxpand if fluid fills part or all of the pleural cavity, if the bronchus is obstructed by secretion or if an acute pneumonitis develops. Accumulation of fluid can best be avoided by mobilization of the adhesions of the superior lobe and "washing" with oxygen. The residual pneumothorax is replaced by oxygen just before closing of the incision. Oxygen stimulates the local defense against infection and its rapid absorption favors the reëstablishment of negative intrathoracic pressure and the reëxpansion of the remainder of the lung. If an empyema develops, the author strongly advises against irrigation with antiseptic solutions as they are of no benefit. Atelectasis of the superior lobe develops generally on the second or third postoperative day and is caused by the tendency of the patient not to cough. Coughing eliminates the mucus and keeps the bronchus open, but is painful for the patient. In this case a curved tube is inserted into the bronchus and permanent suction is maintained. Interstitial pneumonitis is a rare complication and results in a rapid destruction of the affected lobe. The only treatment is removal of the lobe. Sulfanilamide pre- and postoperatively prevents this complication.—*Complicaciones pulmonares en el post operatorio de las lobectomias inferiores, O. L. de Goycoechea, Prensa méd. argent., September 27, 1944, 31: 1930.*—(*W. Swienty*)

Congenital Absence of Lung.—Various theories have been offered to explain agenesis of a lung. The most plausible of them postulates that there is a developmental error of endogenous origin and consequently not only a primary defect in the pulmonary-vascular and respiratory system but also other abnormalities of development. The formation of the lungs from two lung buds on the ventral surface of the esophagus begins in the 3 mm. embryo; therefore, complete absence of a lung is the result of a developmental defect occurring early in fetal life. Three groups can be distinguished: (*1*) true aplasia (no lung, bronchus or blood supply); (*2*) primordial bronchial bud without lung tissue; and (*3*) extreme hypoplasia. In the two latter groups the developmental error occurs at a later stage of embryonic life. The majority of cases reported belong in the first group. Two of the authors' 5 cases fell in the first, 3 in the second group. Agenesis of a lung may be asymptomatic and difficult to discover on physical examination. The space of the absent lung is usually filled with heart, thymus and other mediastinal structures, sometimes also with fluid. Roentgenograms reveal a homogeneous shadow on the affected side, displacement of the heart and mediastinum, elevation of the diaphragm and narrowing of intercostal spaces. In the diagnosis, bronchoscopy and bronchography are essential. The prognosis is guarded, since it depends not only on the effects of the absence of one lung, but also on the possible presence of other serious congenital anomalies. Five cases are reported; 4 of these are living an essentially normal life, while the fifth died of pneumonia.

Other anomalies of the tracheobronchial tree are recognized with increasing frequency as bronchograms become more commonly employed. Five cases illustrative of varying degrees of abnormalities of the tracheobronchial system are presented.—*Congenital Absence of the Lung (Agenesis) and Other Anomalies of the Tracheobronchial Tree, C. F. Ferguson & E. B. D. Neuhauser, Am. J. Roentgenol., November, 1944, 52: 459.*—(*P. Lowy*)

Pulmonary Disease and Mega-Esophagus.—Five cases of pulmonary disease (abscess, bilateral aspiration pneumonitis, diffuse fibrosis and bilateral bronchiectasis) associated with mega-esophagus are reported. It is stressed that mega-esophagus may be nearly or entirely asymptomatic and its presence may be overlooked if respiratory symptoms are in the forefront. On the plain chest roentgenogram, mega-esophagus causes a widening of the mediastinal shadow to the right; in cases of pouch formation in the esophagus the border of the mediastinal shadow may be scalloped or lobulated. Sometimes a fluid level is observed in the dilated esophagus, or a double air column (trachea and esophagus) may be seen overlying the upper dorsal spine. In the lateral view the trachea is displaced anteriorly. When any of these signs are present, studies by means of a contrast medium are called for. The occurrence of the pulmonary diseases mentioned earlier may be explained on the basis of nocturnal regurgitation and aspiration of food particles stagnating in the esophagus. The causal relationship between this pathogenetic mechanism and bronchiectasis is not generally accepted. Since it appears that the association of mega-esophagus and various pulmonary diseases is not merely coincidental, careful investigation of patients with mega-esophagus or with pulmonary disease of unknown etiology is warranted.—*Pulmonary Disease Associated with Mega-Esophagus, H. S. Weens, Am. J. Roentgenol., November, 1944, 52: 472.*—(*P. Lowy*)

Foreign Bodies.—Aspiration of vegetable foreign bodies (peanuts, various types of seeds, etc.) is most common between the ages of 17 and 27 months. When the foreign body reaches the larynx it causes a violent laryngospasm; this is followed by forceful inspiration which draws the foreign body into the tracheobronchial tree. In suspected cases the parents should be questioned as to a history of choking. Roentgenograms are valuable only if they are positive and not misinterpreted. Among physical signs the presence of a wheeze is of the greatest importance; asthmatoid râles, tympanitic percussion note, diminished or absent breath sounds are suggestive. In a certain number of cases the picture of the "drowned lung" rapidly develops and becomes fatal unless the foreign body is removed bronchoscopically. In less fulminating cases emphysema, atelectasis, lung abscess, bronchiectasis or empyema may follow. All these serious consequences can be prevented by early bronchoscopy. Sedatives, narcotics and cough mixtures should not be used if the presence of a foreign body is suspected.—*Vegetable Foreign Bodies in the Tracheobronchial Tree, P. G. Bunker, Journal—Lancet, November, 1944, 64: 369.*—(*P. Lowy*)

Pilocarpin in Diseases of Lung.—Injection of pilocarpin causes a pulmonary vasoconstriction. The chest X-ray film appears clearer and pathological changes become more clearly delimited from surrounding normal pulmonary tissue. Therapeutically, pilocarpin causes decrease of sputum, cough, night-sweats and dyspnea. This drug is particularly valuable in bronchiectasis, but good results may be obtained in pulmonary tuberculosis also if pilocarpin is used as an adjunct to general treatment.—*Die Pilocarpin-behandlung der Lungenerkrankungen, V. Papilian, I. Ursu, N. Mihail & F. Antonescu-Mazilu, Wien. med. Wchnschr., 1942, 1: 259.*—(*G. Simmons*)

Fibrolipoma of Lung.—In the case described the patient suffered for twenty-two years from dyspnea and mucopurulent expectoration every winter. The sputum was always negative for acid-fast bacilli. The sedimentation rate was 76 mm. in one hour. In the X-ray

film the right hemithorax was completely obscured and opaque and there was evidence of cavity formation in the apex. A tentative diagnosis of far advanced pulmonary tuberculosis and bronchiectasis was made. On autopsy the right lung was fibrolipomatous throughout. Only in small and rare areas could normal pulmonary tissue be found. Microscopically, there was a typical lipoma, with infiltration of lymphocytes and plasma cells. Many tuberculous nodules with caseation, calcification and even bone formation were present. This ossification is evidence of progressive metaplasia. A few Langhans cells were found. Many large and small foci of fatty tissue were found throughout the lung. In the authors' opinion, the fat formation is due to the prolonged inflammatory process, and is a sign of temporary or even definite healing of the tuberculous process.—*Fibrolipomatosis pulmonar de sustitucion (a proposito de una observacion), P. I. Elizalde, R. I. Latienda & L. L. Boffi, Rev. Asoc. méd. argent., May 15, 1944, 58: 282.*—*(W. Swienty)*

Bronchopulmonary Tumors.—The differentiation of bronchopulmonary tumors and their early diagnosis is now more important than before, as pulmonary surgery has reached its present height. A group of malignant tumors of the large bronchi is described. They are known as "carcinoids" (or bronchial adenomata), "scar-cancers" and "carcinosarcomata." The pathologico-anatomical findings of one case of each are described. The bronchial adenoma extended into the free lumen of the left main bronchus, which it occluded, and caused the collapse of the lung. Neoplastic cells were arranged in alveoles. There was little anaplasia with rare mitosis. Some cavities invested by the typical ciliated epithelium of the bronchial wall were found. The central zone of the tumor was ossified. All this is evidence that the tumor was originally benign but had suffered malignant transformation. There were no metastases within the lung tissue, but metastases with typical bronchial and alveolar arrangement were found in the liver. The development of the tumor is slow. It is encapsulated and grows by direct invasion. The "scar cancer" was found at the site of an anthracotic subpleural scar. The scar tissue was surrounded by neoplastic cells which invaded the pleura. It is evident that this neoplasm developed in a tuberculous scar and infiltrated the bronchi from there. There were metastases in the thoracic lymph nodes and in the opposite lung. This latter was probably implanted by aspiration. The "carcinosarcoma" developed in the right main bronchus. The tumor was not pedunculated, obstructed the main bronchus completely and penetrated deeply into the trachea. The tumor was surrounded by an exudate of leucocytes. Its main part consisted of sarcomatous tissue which was very richly vascularized. The neoplastic cells were polymorph, fusiform and had vesicular nuclei. On the surface of the tumor small zones of typical squamous carcinoma were found. These are real carcinosarcomata with anaplasia. The primary carcinoma is changed into a sarcoma. —*Contribucion anatomopatoligica al estudio de los tumores broncopulmonares, J. G. Warcalde, Rev. Asoc. méd. argent., September 30, 1944, 58: 793.*—*(W. Swienty)*

Cancer of Lung.—A statistical study was made of 100 cases of histologically proved cancer of the lung. The male-female ratio was more than 7 to 1. The largest number of cases (36) occurred in the sixth decade; the youngest patient was 26 years old, the oldest 78. Cough, fever and weight loss, in this order, were the symptoms most frequently complained of; dyspnea, chest pain, hemoptysis were also common. Fifty-five of the cases were bronchoscoped, and in 39 (71 per cent) of these the tissue removed was reported as carcinoma. Roentgenography, on the other hand, is never absolutely diagnostic, only suggestive. Thirty-two thoracotomies were done, but only 7 radical operations were possible and only 2 of these could be considered successful. Of 7 aspiration biopsies attempted 5 gave positive results, but the procedure is often technically difficult. Roentgenographically, the central type originating in the main

or primary bronchi was the most common; these tumors cause bronchostenosis, with resulting collapse pneumonitis and suppuration distal to it. The other four types, in the order of their frequency, are the following: peripheral, superior sulcus, segmental and diffuse. The average survival time was 245 days. Metastases were most common in the regional lymph nodes, pleura, bones, retroperitoneal lymph nodes and adrenals. Thirty-four cases were given radiotherapy, with symptomatic relief in 10 of them. Fifty per cent of all patients had epidermoid carcinoma, 18 per cent adenocarcinoma, 32 per cent undifferentiated tumor.—*Primary Cancer of the Lungs, K. L. Mitton & N. M. Hardisty, Am. J. Roentgenol., May, 1944, 51: 555.*— (*P. Lowy*)

Tumor of Lung.—During a period of four years 9 patients were seen who were referred to a psychiatrist and/or neurologist since they suffered from violent neuralgic pains with a negative hysterical depression. All patients had a markedly raised blood sedimentation rate. A diagnosis of malignant pulmonary growth was established by radiologic examinations or autopsy findings. The psychologic symptoms and the diffuse neuralgic pains are due to a toxic disorder of the nervous system; localized neuralgic pains may be caused by pressure from the tumor itself or from metastases. The triad of violent neuralgic pains with a negative hysterical depression and an elevated blood sedimentation rate demands an immediate roentgenological examination of the lungs.—*The Initial Neurologic and Psychiatric Syndrome of Pulmonary Growth, A. M. Meerloo, J. A. M. A., October 28, 1944, 126: 558.*—(*H. Abeles*)

Bronchial Carcinoma.—Of 5,515 necropsies performed in the Department of Pathology of the University of Chicago in the forty years from 1902 to 1941, 126, or 2.3 per cent, revealed primary carcinoma of the lung. These constituted 7.6 per cent of all malignant tumors, being fifth in frequency, and 10.3 per cent of carcinomata, being third in frequency. Expressed in terms of percentage of all tumors, primary carcinoma of the lung showed a slight increase in males but not in females over the period studied; the increase was less than that of carcinoma of the colon and intracranial tumors, but greater than that of carcinoma of the pancreas and stomach. When the crude figures were corrected for changes in the annual number of necropsies, for the increase in tumors of all kinds among the necropsy population and for the shift in sex ratio and average age of this same population, it was seen that the increase was apparent rather than real. The only variable which could not be controlled, namely the shift in the type of patient admitted, can easily account for the slight increase noted.—*Increase of Primary Carcinoma of the Lung with Special Reference to Its Increase, P. E. Steiner, Arch. Path., March, 1944, 37: 185.*—(*D. G. Freiman*)

Bronchial Cancer.—In bronchogenic tumors the shadow seen on the roentgenogram is usually larger than the tumor itself, owing to atelectasis distal to the neoplasm. Sometimes, however, emphysema instead of atelectasis is produced. A 44 year old male is described. He was admitted complaining of fever and cough, productive of purulent sputum. An X-ray film of the chest revealed a density in the upper two-thirds of the left lung. The diagnosis was unresolved pneumonia; sulfonamide was given, and a month later the density was smaller. One year later the patient complained of dyspnea, cough, stridor and pain in the left chest. A roentgenogram disclosed a density in the region of the left apex. On bronchoscopy a tumor was found in the left main bronchus; histological examination revealed squamous cell carcinoma. Pressure readings in the left pleural space were plus 8, minus 6. On exploration the left upper lobe was found to be atelectatic, the left lower lobe tremendously emphysematous. The tumor was inoperable. The sequence of events was probably as follows: partial atelectasis of the left upper lobe, complete atelectasis of left upper lobe, obstructive emphysema of left lower lobe.—*Obstructive Phenomena Associated with Primary Bron-*

chial Cancer, W. J. Ozlin, I. A. Bigger & P. P. Vinson, Am. J. Roentgenol., August, 1943, 50: 207.—(P. Lowy)

Bronchial Cancer.—Opinions differ as to the usefulness of roentgen therapy in prolonging the life of patients with bronchogenic cancer. The author treated 167 cases, and compared the survival time with that of 119 untreated cases. The survival time was calculated from the time of making the diagnosis. All cases were microscopically proved. The total dose varied from 1,250 r to 13,750 r; those patients who received more than 2,500 r had more than one course of treatment. The technique of irradiation varied considerably. All but one of the untreated patients died within nine months, none of them was alive after one year. In the treated group, 18 patients lived one year or longer, one being alive after six years. Ninety-four per cent of the untreated and 67 per cent of the treated cases died within six months. It is concluded that roentgen therapy apparently has some life-prolonging effect in bronchogenic cancer, and therefore every patient with inoperable carcinoma of the lung should be given the benefit of this treatment.—*Roentgen Therapy for Bronchiogenic Cancer, B. P. Widmann, Am. J. Roentgenol., January, 1944, 51: 61.—(P. Lowy)*

Diagnosis of Bronchial Cancer.—The search for cancerous cells in the embedded expectoration is a simple and important method in the diagnosis of pulmonary cancer. In 137 cases studied, 32 showed positive findings. Care must be taken to use true expectoration of bronchopulmonary source and fix it immediately. Microscopic diagnosis is based principally on the larger size of the cancerous cells (except in the case of the so-called cancer of small cells), the character of the protoplasm and the pathological changes of the nucleous (increase in size, more marked pigmentation, irregularity, etc.). The latter pathological changes are the most important. Generally cancerous cells are isolated and not in groups, but nevertheless permit diagnosis. Only when the pathology of the cells is typical should the finding be considered positive. Only in this way can the diagnostic errors be kept at a minimum. In 439 cases which were not cases of pulmonary cancer, positive findings were erroneously shown in the expectoration of 6 cases. Therefore the pathologist must study the case on the basis of its clinical findings and repeat the examination in doubtful cases. This experience leads to the belief that the search for cancerous cells in the embedded sputum is a valuable as well as a simple method in the diagnosis of cancer of the lung. This method should, therefore, be used widely in all cases of bronchopulmonary diseases, the etiology of which is not absolutely clear. This should be done even in the cases of young patients, even if there are no clinical symptoms of cancer.—*Importancia de la inclusion de esputos para el diagnostico del cancer broncopulmonar, R. A. Izzo & L. Irigoyen, Publ. d. Centro Investig. Tisiol., 1943, 7: 323.—(H. Behm)*

Plasmocytoma of Lung.—A case of solitary plasmocytoma in the lung of a 30 year old Italian housewife is reported. Following a spontaneous abortion and several weeks of low-grade fever, a mass was discovered in the lung. At the time of admission to the hospital the patient was entirely symptomless. Roentgenographic examination revealed a round, sharply circumscribed, dense shadow about 5 cm. in diameter in the upper and mesial portion of the left pulmonary field. The skull and long bones were negative. The urine was negative for Bence-Jones protein. A left upper lobe lobectomy was performed and was followed by an uneventful convalescence. A well encapsuated, firm, resilient, pearly gray mass was present in the mesial portion of the lobe anterior to the hilar bronchi and vessels. On microscopic examination the predominant cell was indistinguishable from the typical plasmocyte; mitotic figures were rare. Scattered groups of lymphocytes were present, especially just beneath the capsule, and clusters of foamy macrophages were also seen. Moderate numbers of acido-

phile hyaline droplets (the so-called Russell bodies) were found throughout. The site of origin of the tumor was not determined.—*Plasmocytoma of the Lung, J. Gordon & G. Walker, Arch. Path., March, 1944, 37: 222.*—(*D. G. Freiman*)

Carcinoma of Trachea.—A 61 year old male lecturer suffered from frequent episodes of coughing with blood-streaked sputum and fatigue over a period of three years. When he developed wheezing which reduced his voice to a whisper he sought medical care. Bronchoscopic examination revealed a tumor just above the carina which extended over the whole cartilaginous portion of the trachea. A biopsy was taken and it was reported as primary carcinoma of the trachea but the possibility of neoplastic thyroid tissue could not be ruled out. Examinations with radioactive iodine ruled out the presence of normal thyroid tissue in the area from which the tumor originated. However, the lack of storage of radioactive iodine in the tumor did not exclude a carcinoma of the thyroid. In five bronchoscopic sessions as much tumor as possible was removed. This was followed by treatment with radium in a special applicator placed through the bronchoscope. Two years after the treatment the patient was well; there was no subjective or objective evidence of tumor or metastases. The only finding was a little thinning of the mucous membrane in the area previously occupied by the tumor.—*Primary Carcinoma of the Trachea; Treatment with Intratracheal Radium; Radioactive Iodine Fails to Show Thyroid Origin, P. H. Pierson, J. A. M. A., September 23, 1944, 126: 206.*—(*H. Abeles*)

Mediastinal Tumors.—The preoperative classification of mediastinal tumors has proved difficult or impossible, but has become increasingly important. Forty-four cases of proved mediastinal tumors were reviewed to determine, if possible, whether any of these tumors had characteristic roentgenological features. It was found that with present methods of X-ray examination no exact differentiation is possible. However, certain diagnostic features have been demonstrated. Bronchogenic cysts are round or ovoid tumors located anywhere within the mediastinum without evidence of calcification or bone erosion. Dermoid cysts and teratomata appear as slightly more irregular masses located in the anterior mediastinum which may contain areas of calcification. Neurogenic tumors are usually smooth in outline, homogeneous in density and may be located in any portion of the mediastinum. Bone erosion is commonly associated with this type of tumor. The outstanding finding in intrathoracic goitre is its attachment to the trachea, often with associated displacement of the latter. Parathyroid and thymic tumors may be recognized as mediastinal tumors, but their character is largely dependent upon other roentgenological or clinical characteristics. Diagnostic pneumothorax might help to determine the location of a tumor, and a trial dose of X-ray may also aid in suggesting the final diagnosis. Careful fluoroscopic study and postero-anterior and lateral films should, of course, be obtained.—*Roentgenologic Features of Mediastinal Tumors, L. L. Robbins, Radiology, August, 1944, 43: 115.*—(*G. F. Mitchell*)

Mesothelioma of Pleura.—A 25 year old woman was admitted to the hospital with a six months' history of lassitude and pain over the left shoulder. Shortly before admission she developed pain in the left chest anteriorly, unaffected by breathing. Physical examination revealed shift of the mediastinum to the right, and, on the left, absent vocal fremitus, flat percussion note from apex to base posteriorly, and bronchial breath sounds. There was marked clubbing of fingers and toes. Clear yellow fluid, negative for tubercle bacilli and tumor cells, was aspirated from the left chest. Dyspnea, not present at the onset of illness, became progressively more severe, and the patient eventually died in respiratory failure. Autopsy revealed a compressed left lung entirely surrounded by a thick, firm gray sheet of tumor displacing it upward and medially. The tumor covered the entire

parietal surface of the left chest wall and was adherent to the diaphragm and pericardium. On section the tumor was gray-white with scattered tiny cystic areas filled with mucoid material; the consistency was almost cartilaginous. Small plaques of tumor were found on the right visceral pleura and around the hilar portion of the right pulmonary artery; tumor nodules were also present in the right lung and over the parietal and visceral surfaces of the peritoneum. Histologically, the tumor was relatively acellular and avascular. In scattered areas cells were seen with nuclei of varying shape and eosinophilic cytoplasm with indistinct outline; the stroma was eosinophilic and fibrillar, arranged in an interlacing and whorled pattern, and rich in reticulum. Signet ring cells were present in moderate numbers and were most numerous in the collagenous portions of the tumor; fat and mucicarmine stains were negative, suggesting that these cells were degenerative rather than secretory. The most striking feature of the tumor was the presence of an atypical osteoid matrix in the central portions, a finding which does not appear to have been previously reported in a case of pleural mesothelioma.—*Mesothelioma of the Pleura, A. V. Postoloff, Arch. Path., April, 1944, 37: 286.*—(*D. G. Freiman*)

Hodgkin's Disease.—Of 55 proved cases of Hodgkin's disease intrathoracic involvement was found in 35 (63 per cent). The latter were classified into five types: mediastinal, parenchymal, pleural, osseous and cardiac. In all these types the character of the disease is determined by the location and the degree of involvement of the lymphoid tissue. Fifty per cent of the cases belonged in the mediastinal group in which discrete or confluent enlargement of the mediastinal lymph nodes is seen roentgenologically. Involvement of the lung parenchyma occurred in 40 per cent of the series. In these cases the differential diagnosis from tuberculosis, pneumonia, bronchogenic carcinoma, sarcoidosis, abscess and metastatic tumor may be difficult. The invasion of the lung parenchyma may occur from the mediastinal nodes, producing the picture of a solid growth, or the infiltration may spread along the peribronhcial and perivascular lymphatics in which case linear densities are seen extending into the lungs from the hilum. In other cases the disease extends through the alveoli, thereby producing a granulomatous consolidation of lobar or lobular character. Nodular lesions are uncommon and were not observed in this series. Bronchogenic carcinoma is simulated if invasion of the bronchi causes bronchial obstruction and atelectasis, while necrosis followed by cavitation raises the question of tuberculosis, lung abscess or carcinoma. The clinical symptoms and signs of parenchymal disease (cough, chest pain, fever, night sweats, weight loss) are not diagnostic. In pleural involvement nodular or infiltrating masses are present on the pleura, usually associated with serous effusion. Involvement of the bones forming the thorax results from direct extension from lymphoid tissue; occasionally the osseous lesions are primary. Rarely, infiltrations extend from adjacent structures into the heart. Twenty-three of the 35 cases of intrathoracic Hodgkin's disease were given radiotherapy, and 17 of these showed definite improvement.—*Intrathoracic Hodgkin's Disease, S. E. Wolpaw, C. S. Higley & H. Hauser, Am. J. Roentgenol., October, 1944, 52: 374*—(*P. Lowy*)

Cystic Fibrosis of Pancreas.—Pulmonary changes (bronchitis, bronchopneumonia, bronchiectasis, atelectasis) are present in the great majority of patients with cystic fibrosis of the pancreas. They are probably due to vitamin-A deficiency resulting from faulty fat absorption; according to another view, the pulmonary picture is based on a coexisting congenital abnormality of the pancreas and lungs. Absence of pancreatic enzymes in the duodenal contents, steatorrhea and low vitamin-A absorption curve are the salient diagnostic features of the disease. The vitamin-A deficiency produces epithelial damage and infection in the lungs; roentgenologically, there are increased, mottled densities in

the hilar regions and streaking and mottling towards the periphery. The consequences of infection are atelectasis and bronchiectasis. Although the pulmonary changes are strikingly similar in cases of cystic pancreatic disease, yet they are not in themselves diagnostic. Similarly, hypomotility associated with dilatation of the small intestine is suggestive but not pathognomonic of the disease.—*Pulmonary Changes in Chronic Cystic Pancreatic Disease, G. J. Baylin, Am. J. Roentgenol., September, 1944, 52: 303.—(P. Lowy)*

Spontaneous Paralysis of Diaphragm.—The author reviews the literature on spontaneous paralysis of the diaphragm. A few are congenital, most are acquired. Inflammatory processes of the hilar lymph nodes and cardiovascular diseases may cause pressure and atrophy of the phrenic nerve and so a paralysis of the diaphragm. The same is true for degenerative process of the muscle or of the pleural covering of the diaphragm, intestinal tumors or a change in the equilibrium between thorax and abdomen. The most common cause is tuberculosis. A systematic X-ray examination would show a far greater number of diaphragmatic paralyses than is generally conceived. The hilar nodes must be very large if they cause pressure on the phrenic nerve. In the author's opinion, this should not be very frequent, especially on the left, as the left phrenic nerve passes far in front of the hilar nodes. He believes that there is in reality a paralysis of the muscle which occurs as a defensive reaction of the diseased hemithorax and has no relation to the enlarged hilum. The transmission of a motor reflex stimulated by the viscera may be the reason for the paralysis. This would explain diaphragmatic paralysis in cases of tuberculosis with only slight enlargement of the tracheobronchial nodes. Pressure on the sympathetic ganglia and the cervical plexus by tuberculous nodes is also of importance. In the review of the etiology, poliomyelitis, hysteria, syphilis, diabetes, alcoholism, rheumatism and gout as well as trauma are of importance. The symptoms of paralysis of the diaphragm are dyspnea and polypnea, asphyxia, difficult, low speech and the sensation of the abdominal viscera moving into the thorax. The X-ray picture shows a raise of the dome of the diaphragm, diminished transparence of the lung tissue, blotting of the costodiaphragmatic angle, paradoxical movement of the diaphragm and shifting of the mediastinum to the non-affected side. A transitory paralysis of the diaphragm may be mistaken for pleurisy or pneumonia. The author describes one case.—*Contribucion al estudio de la paralisis diafragmatica espontanea, C. W. Grobli, Prensa méd. argent., September 6, 1944, 31: 1764.—(W. Swienty)*

Hemidiaphragmatic Paralysis.—A man was seen in whom a paralysis of the medial and anterior portion of the right hemidiaphragm was present as a remainder of an old anterior poliomyelitis. Neurological examination revealed that the left side of the cervical cord was more affected than the right; apparently there was a crossing of the phrenic nerves in the spinal cord. Scoliosis to the contralateral side and considerable damage of respiratory reserve were found.—*Partial Paralysis of a Hemidiaphragm, H. Abeles & G. C. Leiner, Am. J. Roentgenol., May, 1944, 51: 572.—(P. Lowy)*

Congenital Absence of Ribs.—Complete absence of ribs may be divided into three groups: (*1*) absence of one rib unilaterally or bilaterally; (*2*) aplasia of two or more ribs unilaterally; (*3*) a miscellaneous group. Any of these categories may be associated with anomalies of the vertebral body. Illustrative cases of each category are recorded. The embryology of the rib and vertebral body is presented. The theories of etiology of complete aplasia of ribs are discussed. (Author's Summary)—*Congenital Absence of Ribs, B. N. E. Cohn, Am. J. Roentgenol., November, 1944, 52: 494.—(P. Lowy)*

Cardiac Arrythmia.—In the evaluation of cardiac arrythmia in pulmonary disease, anatomically adjacent factors should be con-

sidered. Otherwise they are significant of the problem of cardiac arrythmias in general. At times they constitute the sole evidence of cardiac disease, but often occur in the absence of such disease. One is justified in correlating their occurrence with existing extracardiac factors, and the clinician may be influenced in his prognosis and therapeutic approach by consideration of this etiologic dependence. In pulmonary tuberculosis, extracardiac factors that may produce abnormal cardiac rhythm may be (*1*) the influence of tuberculosis on the intravascular pulmonary tension, (*2*) involvements of pericardium and myocardium from adjacent pulmonary tissue, (*3*) toxic influences of the disease upon the heart, (*4*) mechanical involvement of the heart and great vessels due to torsion and displacement and (*5*) reflexes due to disturbed anatomy and physiology. The electrocardiograms of 1,000 cases admitted to Sea View Hospital are reviewed. In 78 there were arrythmias; 12 of these were sinus arrythmia, 22 were premature auricular contraction and ventricular contraction, 14 were various types of auriculoventricular conduction impairment, mostly first stage heart block, 20 were intraventricular conduction impairment and 10 were miscellaneous arrythmias. When tabulated according to disease, it was noted that there were four times as many cases of arrythmia in the active as in the inactive group. When studied in relation to age and cardiac disease, it was found that sinus arrythmias occurred in the younger age group with one exception, premature contractions were equally divided in age groups, and most had evidence of cardiac disease. In the group with miscellaneous arrythmias there was evidence of cardiac disease in all but one and most were in the older age group. Intraventricular and auriculoventricular conduction impairments predominated in those with associated cardiac disease. Evidence of cardiac disease consisted of objective criteria, for example, history of syphilis or rheumatic fever, physical findings such as peripheral sclerosis or unmistakable cardiac murmurs indicative of mitral or aortic disease, X-ray evidence of cardiac enlargement, and typical electrocardiogram changes indicative of coronary artery occlusion. Most of those showing the auriculoventricular block occur in the younger group, but in only 3 was there no evidence of cardiac disease other than an abnormal electrocardiogram. All 3 (aged 48, 18 and 32) had a mediastinal shift. It is possible that mediastinal displacement can alter the configuration of the P wave producing a picture of first-stage heart block, but it is also possible that toxic influences may play a rôle. The effect of mediastinal displacement on the irritability of the myocardium has been stressed by a number of foreign writers, and Brumfield in this country reported 7 with extrasystoles in 15 cases of mediastinal distortion. There may be a higher percentage in this group (22) as only one electrocardiogram was made, but only one per cent of the group gave no evidence of intrinsic heart disease. This incidence is small compared to the number with mediastinal distortion seen at Sea View. As far as is known only one other large series of pulmonary tuberculosis cases thus studied has been reported. While evidence points to intrinsic cardiac disease as the etiologic factor in producing arrythmias, the results suggest that extracardiac factors may play a rôle in precipitating these arrythmias. The side of involvement seems to be no factor. It is conceivable that where there is mediastinal torsion, the relationship and course of the cardiac nerves may be disturbed producing increased irritability of the myocardium. The authors conclude that pulmonary tuberculosis does not affect the incidence of abnormal cardiac rhythm or abnormalities in the conduction mechanism and that in most cases other clinical or laboratory evidence of intrinsic cardiovascular involvement can be elicited. However, right pulmonary lesions involving the mediastinum may constitute an extracardiac factor in the causation of the arrythmias and conduction abnormality in a previously diseased myocardium.—*Cardiac Arrythmias in 1000 Cases of Pulmonary Tuberculosis, T. T. Fox & A. L. Bobb, Am. J. M. Sc., August, 1944, 208: 201.*—(*G. F. Mitchell*)

Hemoptysis in Mitral Stenosis.—In mitral stenosis large hemoptyses can occur the source of which is not understood. A method for injecting the bronchial veins of lungs, fresh from autopsies, is described. This method indicates the presence of direct venous connections between the bronchial and pulmonary veins in men of all ages. Mitral stenosis causes dilatation of the bronchial veins in the submucosa of the larger bronchi as a result of the establishment of a collateral flow through them. In cases of mitral stenosis in which infarction and acute pulmonary edema are not present, hemoptysis is probably due to bleeding from these dilated veins. Age, hypertension, and arteriosclerosis do not affect the bronchial venous bed, but some dilatation occurs in chronic congestive heart failure of long standing, although the only lesion of the mitral valve may be dilatation of the valve ring.— *Varices of the Bronchial Veins as a Source of Hemoptysis in Mitral Stenosis, F. C. Ferguson, R. E. Kobilak & J. E. Deitrick, Am. Heart J., October, 1944, 28: 445.—(G. C. Leiner)*

Anomaly of Pulmonary Artery.—An autopsy finding offered the opportunity of studying a rare vascular anomaly in the wall of the left pulmonary artery in a female who, though she was 88 years old, had no other vascular alterations in the lesser circulation, and died of postoperative peritonitis after a colectomy. Opening the artery lengthwise near the first branching, an engorged curved vessel was seen clearly outlined through the endothelial lining, 20 mm. long, with two small branches in the convex side. No other analogous arrangement was found in the rest of the artery. The vessel, raising the inner lining, was located in the intima and serial sections revealed that, in some places, it was in the *tunica media*. There was also a vascular net with vessels of different calibres and structure, ranging from the arterial type to ample canals, that was traced through the different layers of the artery and was connected directly with the *vasa vasorum* and with the lumen of the artery, opening by means of ostial orifices in the intima, supplied with valve-like flaps. This fact suggested that the blood-stream ran from the vasa vasorum toward the lumen of the artery. The absence of inflammatory or other leisons that could explain the presence of this arrangement suggested that it was congenital. —*Raro dispositivo vascular en la pared de la rama izquieda de la arteria pulmonar, D. Branchetto-Brian & E. F. Lascano, Rev. Asoc. méd. argent., March, 1944, 58: 105.—(J. Badell)*

Adrenal Amyloidosis.—During the past eleven years amyloidosis of the adrenals was seen in 354 cases on autopsy at Sea View Hospital. Amyloidosis of the adrenal increases the consistency, size and weight of the organ. The zona fasciculata is involved first, the zona glomerulosa last. In none of the cases was there clinical evidence of Addison's disease. An attempt was made to find signs of adrenal insufficiency: The salt restriction test shows —in the absence of edema—fairly close correlation between the amount of chloride excreted and the extent of amyloidosis. Blood pressure, changes in renal function and carbohydrate metabolism, adynamia and gastrointestinal disturbances are of little or no assistance. In severe amyloidosis of the adrenals the body temperature is usually only slightly elevated or even normal in spite of active disease.—*Adrenal Amyloidosis, Marguerite G. Stemmerman & O. Auerbach, Arch. Int. Med., November, 1944, 74: 884.—(G. C. Leiner)*